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     (FILE 'HOME' ENTERED AT 14:00:37 ON 13 DEC 2004)

     FILE 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
     AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOENG, BIOSIS, BIOTECHDS, BIOTECHNO,
     CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU,
     DDFB, DGENE, DISSABS, DRUGB, DRUGMONOG2, ...' ENTERED AT 14:00:47 ON 13
     DEC 2004
L1            301 SEA (IRON)(P)(URIN?)(P)(GLOMERULONEPHRITIS OR NEPHROPATHY OR
                  SEGMENTAL SCLEROSIS OR ERYTHEMATOISIS OR HEMOLYTIC UREMIC OR
                  HENOCH-SCHONLEIN)
L2             17 SEA (IRON)(8W)(URIN?)(20W)(GLOMERULONEPHRITIS OR NEPHROPATHY
                  OR SEGMENT SCLEROSIS OR HEMOLYTIC UREMIC OR HENOCH-SCHONLEIN)
L3             10 DUP REM L2 (7 DUPLICATES REMOVED)
                  D IBIB ABS L3 1-10
L4            140 SEA (IRON)(P)(URIN?)(P)(NEPHROTIC OR NEPHROSIS)
L5              8 SEA (IRON)(8W)(URIN?)(15W)(NEPHROTIC OR NEPHROSIS)
L6              3 DUP REM L5 (5 DUPLICATES REMOVED)
                  D IBIB ABS L6 1-3
L7           1254 SEA (DEFERIPRONE OR DEFEROXAMINE OR POLYANIONIC OR POLYAZA)(P)(
                  KIDNEY OR GLOMERULONEPHRITIS OR NEPHROPATHY OR SEGMENTAL
                  SCLEROSIS OR ERYTHEMATOISIS OR HEMOLYTIC UREMIC OR HENOCH-SCHON
                  LEIN OR NEPHROTIC OR NEPHROSIS)
L8            156 SEA (DEFERIPRONE OR DEFEROXAMINE OR POLYANIONIC OR POLYAZA)(15W
                  )(KIDNEY DISEASE OR KIDNEY OR NEPHROTIC OR NEPHROSIS OR
                  GLOMERULONEPHRITIS OR NEPHROPATHY)
L9             81 DUP REM L8 (75 DUPLICATES REMOVED)
                  D IBIB ABS L9 1-81
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FIELD CODE - 'AND' OPERATOR ASSUMED '(IRON)(P)(URIN?'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'URIN?)(P)(NEPHROTIC'
L4 140 (IRON)(P)(URIN?)(P)(NEPHROTIC OR NEPHROSIS)

=> s (iron)(8w)(urin?)(15w)(nephrotic or nephrosis)
43 FILES SEARCHED...
L5 8 (IRON)(8W)(URIN?)(15W)(NEPHROTIC OR NEPHROSIS)

=> dup rem l5
DUPLICATE IS NOT AVAILABLE IN 'ADISINSIGHT, ADISNEWS, BIOCOMMERCE, DGENE, DRUGMONOG2, FEDRIP, FOREGE, GENBANK, IMSPRODUCT, IMSRESEARCH, KOSMET, MEDICONF, NUTRACEUT, PCTGEN, PHAR, PHARMAML, PROUSDDR, RDISCLOSURE, SYNTHLINE'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING COMPLETED FOR L5
L6 3 DUP REM L5 (5 DUPLICATES REMOVED)

=> d ibib abs l6 1-3

L6 ANSWER 1 OF 3 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 1
ACCESSION NUMBER: 1995:260583 BIOSIS
DOCUMENT NUMBER: PREV199598274883
TITLE: Non-iron mediated alteration in hepatic transferrin gene expression in the nephrotic rat.
AUTHOR(S): Kaysen, George A. [Reprint author]; Sun, Xihua; Jones., Hardin, Jr.; Martin, Victor I.; Joles, Jaap A.; Tsukamoto, Hidekazu; Couser, William G.; Al-Bander, Hamoudi
CORPORATE SOURCE: Div. Nephrol., Univ. California Davis, TB 136, Davis, CA 95616, USA
SOURCE: Kidney International, (1995) Vol. 47, No. 4, pp. 1068-1077. CODEN: KDYIA5. ISSN: 0085-2538.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 13 Jun 1995
Last Updated on STN: 13 Jun 1995

AB Both transferrin and the **iron** it carries are lost in the **urine** in the **nephrotic** syndrome. Patients may develop hypochromic microcytic anemia and synthesis of transferrin, a protein regulated in large part by iron availability, is increased. Transferrin synthesis has also been reported to be increased in liver slices from rats with hereditary analbuminemia, and their plasma transferrin levels are increased, suggesting that transferrin synthesis may be stimulated by processes other than iron depletion in this hypoalbuminemic condition. Transferrin metabolism was studied in rats with Heymann nephritis (HN), in a strain of Sprague-Dawley (SD) rats with hereditary analbuminemia (Nagase analbuminemic rats (NAR)), and in normal SD rats. Plasma transferrin concentration and mass was decreased significantly in HN, but increased in NAR. Transferrin synthesis was increased both in NAR (measured either as the disappearance of (125I) labeled transferrin or as the incorporation of (3H) phenylalanine) and in HN (incorporation of (3H) phenylalanine). The fractional rate of transferrin catabolism was unchanged in NAR. Thus transferrin mass was increased in NAR entirely as a consequence of increased synthesis. Transferrin and albumin synthesis correlated with one another in both HN and SD (P lt 0.001). Transferrin mRNA was increased in both HN and NAR and was unaffected by administration of iron to HN. Hepatic transferrin and albumin mRNA levels were also correlated positively in HN and SD, suggesting that increased hepatic synthesis of both proteins might be responding to the same stimuli. Transferrin gene transcription was increased in both HN and NAR and was unaffected by administration of iron to HN. Transferrin mRNA was not increased in the testis in either HN or NAR, suggesting that augmentation in transferrin gene expression is driven by a non-iron dependent process and is confined to the liver.

ANSWER 3 OF 4 JICST-EPlus COPYRIGHT 2004 JST on STN
AN 910796543 JICST-EPlus
TI Studies on the pathogenesis in iron deficiency anemia. Part 1. Urinary iron excretion in iron deficiency anemia patients and rats in various iron states.
AU NAKANISHI NORIHIKO
CS Okayama Univ., School of Medicine
SO Okayama Igakkai Zasshi, (1991) vol. 103, no. 7-8, pp. 803-811. Journal Code: Z0158B (Fig. 3, Tbl. 2, Ref. 22)
ISSN: 0030-1558
CY Japan
DT Journal; Article
LA Japanese
STA New
AB In the "iron excretion test", urinary iron excretion after injection of saccharated iron oxide has been reported to be accelerated in relapsing idiopathic iron deficiency anemia. To determine the relevance of urinary iron excretion to clinical factors other than iron metabolism, 15 clinical parameters were **evaluated**. The serum creatinine level was positively and the serum albumin level was negatively correlated with urinary iron excretion, showing coefficients of r=0.97,-0.86 respectively, and suggesting a relationship between urinary iron excretion and subclinical abnormalities of kidney function. In experimental studies, the relation of urinary iron excretion to the renal function was examined by administration of iron in various forms to rats. Only saccharated iron oxide was excreted; chondroitin sulfate Fe, Tf-Fe and ferric chloride were not excreted in the urine. Then, iron excretion was examined in iron deficient, iron overloaded and puromycin aminonucleoside(PA)-treated animals. Iron deficient rats did not show any change in urinary iron excretion compared to the controls. Urinary iron excretion was increased in iron overloaded rats, and was further increased in the PA-treated group. These findings suggest that the subclinical abnormality in **kidney** function leads to the increased **urinary iron** excretion as a possible factor in the pathogenesis of relapsisg iron deficiency. (author abst.)
CC EJ03010L; EJ02036Y (591.11.05; 591.13 OTHERS)
CT iron deficiency anemia; pathogenicity; pathophysiology; iron; metabolic error; urinary excretion; chalybeate; iron oxide; iron chloride; transferrin; chondroitin sulfate; rat
BT nutrition disorder; disorder/trouble/obstacle; metabolic disease; disease; anemia; hematologic disease; property; fourth row element; element; iron group element; transition metal; metallic element; anomaly; excretion; hematinic; hematological drug; drug; mineral preparation; metabolic drug; metal oxide; oxide; chalcogenide; oxygen group element compound; oxygen compound; iron compound; iron group element compound; transition metal compound; chloride; chlorine compound; halogen compound; halide; blood protein; blood component; component; animal protein; protein; chromoprotein; carboxamide; glycosaminoglycan; mucopolysaccharide; amino sugar; carbohydrate; polysaccharide; aliphatic carboxylic acid; carboxylic acid; pyranoside; glycoside; polyuronide; inorganic acid ester; ester; sulfuric acid derivative; sulfur oxyacid derivative; sulfur compound; Myomorpha; Rodentia; Mammalia; Vertebrata; animal

ANSWER 1 OF 10 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 1996:758843 CAPLUS
DOCUMENT NUMBER: 126:102469
TITLE: Increased urinary iron excretion rate in patients with non-insulin dependent diabetes mellitus
AUTHOR(S): Nishiya, Koji; Takamatsu, Kazunaga; Yoshimoto, Yukio; Ikeda, Yukio; Ito, Hiroyuki; Hashimoto, Kozo
CORPORATE SOURCE: 2nd Dep. Intern. Med., Kochi Med. Sch., Nankoku, 783, Japan
SOURCE: Rinsho Byori (1996), 44(12), 1201-1202
CODEN: RBYOAI; ISSN: 0047-1860
PUBLISHER: Rinsho Byori Kankokai
DOCUMENT TYPE: Journal
LANGUAGE: Japanese

AB Urinary iron excretion rate (u-FeER, μg/min) in urine at night time from 58 patients with non-insulin dependent diabetes mellitus and 8 controls was measured by atomic absorption. The patients were divided into 3 groups according to urinary albumin excretion rate (u-AER), namely, group I (n = 44):less than 20 μg/min of u-AER; Group II (n = 9): 20 ≤ u-AER < 200 μg/min; Group III (n = 5): more than 200 μg/min of u-AER. The u-FeER in group II (56.3 ± 14.8 ng/min, mean ± SEM) was significantly higher than that in controls (4.0 ± 1.6 ng/min), group I (8.3 ± 1.6 ng/min) and group II (18.5 ± 6.5 ng/min). The increase of u-FeER in group III indicates that urinary iron may at least partly play a role in development of diabetic nephropathy.

ANSWER 6 OF 10 CABA COPYRIGHT 2004 CABI on STN DUPLICATE 3

ACCESSION NUMBER: 77:68578 CABA
DOCUMENT NUMBER: 19761446500
TITLE: Transferrin loss into the urine with hypochromic, microcytic anemia
AUTHOR: Hancock, D. E.; Onstad, J. W.; Wolf, P. L.
CORPORATE SOURCE: Dep. Pathology AID 7200, University Hospital, 225 West Dickinson, San Diego, Calif. 92103, USA.
SOURCE: American Journal of Clinical Pathology, (1976) Vol. 65, No. 1, pp. 73-78.
ISSN: 0002-9173
DOCUMENT TYPE: Journal
LANGUAGE: English
ENTRY DATE: Entered STN: 19941101
Last Updated on STN: 19941101

AB A Mexican boy 7 years old with the nephrotic syndrome acquired microcytic, hypochromic anaemia, secondary to heavy loss of Fe and transferrin in urine. Serum Fe was 12 mu g/100 ml and serum iron-binding capacity 12 mu g/100 ml; Fe in urine was 64 mu g/100 ml and **iron**-binding capacity of **urine** 366 mu g/100 ml. Renal biopsy indicated **glomerulonephritis**. Examination of proteins in serum and urine is recommended for patients with nephrosis.

☐ 1: Am J Kidney Dis. 1995 Feb;25(2):314-9.

Related Articles, Li

Am J Kidney Dis

Urinary iron speciation in nephrotic syndrome.

Cooper MA, Buddington B, Miller NL, Alfrey AC.

Department of Medicine, Veterans Administration Medical Center, Denver, CO.

In nephrotic syndrome, iron is presented to the tubule fluid in a nonreactive form in association with transferrin as a result of the glomerular protein leak. At an alkaline pH, iron remains bound to transferrin throughout the nephron and is excreted as such in the urine. As urine pH decreases below 6, iron is dissociated from transferrin. In the dissociated form, iron exists in the urine i soluble, ultrafiltrable, and labile state. It is suggested that iron is maintained i this state by chelation to a relatively small organic compound, such as citrate This non-transferrin-bound iron is capable of catalyzing bleomycin degradati of DNA, suggesting that this labile form of iron is able to catalyze free radica formation and cause tubule cell injury. Urine from proteinuric states represer one of the few, if not only, biologic fluids containing large amounts of reacti iron species. This may explain the mechanism by which proteinuric states cause tubulointerstitial disease and renal failure.

PMID: 7531396 [PubMed - indexed for MEDLINE]

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Urinary iron speciation in nephrotic syndrome. Am J Kidney Dis. 1995 Feb;25(2):314-9.

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Department of Medicine, Veterans Administration Medical Center, Denver, CO.

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PMID: 7531396 [PubMed - indexed for MEDLINE]

Renal iron handling in the nephrotic syndrome.

Kidney Int. 1990 Jun;37(6):1409-13.

Alfrey AC, Hammond WS.

Department of Medicine and Pathology, Veterans' Administration Medical Center, Denver, Colorado.

Renal iron handling was characterized in three experimental models of the nephrotic syndrome: puromycin aminonucleoside, adriamycin and nephrotoxic serum. In adriamycin-induced nephrotic syndrome, which has previously been shown to result from alterations in pore size of the filtration barrier, the transferrin leak was most severe with a fractional clearance of 25%, a value identical to albumin. In contrast, in puromycin nephrotic syndrome and nephrotoxic serum nephritis the fractional clearance of transferrin was never greater than 2% and consistently less than the fractional clearance of albumin. The fact that iron/transferrin ratios in urine and serum were frequently different, sometimes higher other times lower, documents that iron and transferrin can be dissociated in tubule fluid and handled differently in regards to tubule uptake. Kidney iron concentration is also increased in both immunological and non-immunological forms of nephrotic syndrome. In the proximal tubule iron is present largely on the luminal aspect of the cell. In contrast, the major deposition of iron occurs in the lysosomes of the

distal tubule cells. Kidney iron concentration does not correlate with tubule fluid iron content but can be prevented from increasing by systemic iron and/or transferrin depletion. This suggests that iron enters the distal tubule cells with transferrin via its receptors from the basolateral side of the distal tubule cells. In association with the increase tubule fluid and kidney iron, there is a marked reduction in kidney selenium and copper content. It is concluded that urinary iron and transferrin losses can vary greatly in different types of experimental renal diseases, and that iron and transferrin can be dissociated in the tubule fluid.(ABSTRACT TRUNCATED AT 250 WORDS)

PMID: 2362400 [PubMed - indexed for MEDLINE]

Postgraduate Medical Journal, 1984, Vol 60, 125-128

ARTICLES

Urinary iron loss in the nephrotic syndrome--an unusual cause of iron deficiency with a note on urinary copper losses

EA Brown, B Sampson, BR Muller and JR Curtis

Two patients with long-standing nephrotic syndrome are described in whom urinary iron losses may have contributed towards an iron deficiency state. Seven other nephrotic patients were also studied. Increased urinary iron excretion was found in six out of nine patients and increased urinary copper excretion in all eight patients in whom it was measured. Trace metal losses in the urine in nephrotics may be important clinically.